PATTERN OF PRIMARY LUNG CANCERS

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CERTIFICATE

This is to certify that the dissertation titled **"PATTERN OF PRIMARY LUNG CANCERS "** submitted by **Dr.BUVANESWARI.D** to the Faculty of General Medicine, The Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.D. Degree (General Medicine) is a bonafide research work carried out by her under our direct supervision and guidance.

Dr.M.KAMARAJ M.D.,

Addl.Professor of Medicine
Chief III Medical Unit,
Department of Medicine,
Madurai Medical College,
Madurai.

Dr. A. AYYAPPAN M.D.,

Professor and Head
Department of Medicine,
Madurai Medical College,
Madurai.

DECLARATION

I, **Dr. BUVANESWARI.D** , solemnly declare that the dissertation titled "**PATTERN OF PRIMARY LUNG CANCER- GRH STUDY"** has been prepared by me.

This is submitted to the **Tamilnadu Dr. M.G.R. Medical University**, Chennai in partial fulfillment of the rules and regulations for the M.D. Degree Branch I (General Medicine).

Place : Madurai

Date : **Dr. BUVANESWARI.D**

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CONTENTS

INTRODUCTION

Lung cancer was considered to be rare in the beginning of the century but has now reached almost epidemic proportions. It is the leading cause of cancer deaths in developed countries and is also rising at alarming rates in developing countries. It is the leading cause of cancer mortality in most of the countries in the world. It remains the most lethal form of cancer in men and has now surpassed breast cancer in women as well in USA, where 170,000 new cases are diagnosed per year.

The Indian subcontinent in South Asia occupies 2.4% of the world land mass and is home to 16.5% of the world population. India is a vast rural panorama with 70% of the population residing in villages in rural surroundings. The worldwide incidence of lung cancer is 14% whereas it constitutes 6.8% of all cancers in India.[10] Non-small cell lung cancer accounts for nearly 85% and small cell lung cancer accounts for 15% to 20% of cases. There are important differences in the clinical spectrum of lung cancer patients in India compared to those in the West. Both the mean and peak ages of lung cancer are lower. The smoker: non-smoker ratios have been lower in most of the Indian studies as compared to those in the West. Though there is increased awarness of hazards of smoking in

developed countries that shows some decline in smoking habits, in India lack of knowledge and awareness of hazards of smoking actually increases the number of smokers and thereby increases the incidence of lung cancer.

Despite advances in imaging techniques and treatment modalities, the prognosis of lung cancer remains poor, with a five-year survival of 14% in early stages and less than 5% in locally advanced stages. Unfortunately only 20-30% of patients present with an operable disease, while most of the patients present in an advanced stage II and III. The main reason for late presentation in our country is the poor health awareness, delayed recognition and the poor referral of patients to the specialised centers.

Almost all patients present with symptoms of lung cancer that they were found to be in an advanced stage of lung cancer. Screening for lung cancer is still a controversy worldwide, is not feasible in our country. And especially in tertiary Government hospital set up, where every patient are only poor, screening could not be done because of their poor affordability.

The present study was undertaken to analyse the clinical presentation, radiological pattern and pathological characteristics of lung cancer at Government Rajaji Hospital, Madurai.

AIMS AND OBJECTIVES

- To study the clinical pattern of lung cancer, with special emphasis to study the most common clinical presentation.

- To study through the clinical presentation, the extent of disease.

- To confirm the diagnosis and to stage them with Imaging, Bronchoscopy and Biopsy.

- To study about the distribution of lobe pattern involved in primary lung cancer.

REVIEW OF LITERATURE

HISTORY

Some 150 years ago, it was an extremely rare disease. In 1878, malignant lung tumors represented only 1% of all cancers. By 1918, the percentage had risen to almost 10% and by 1927 to more than 14%[7]. The smoking of cigarettes had become popular shortly before the turn of the century. Originally, cigarettes were hand rolled and this made them expensive. In 1876, the cigarette manufacturer Allen & Ginter offered a prize for the development of a machine that would speed up the process. James Albert Bonsack developed a machine that could make 70,000 cigarettes in a 10 h day. World War I helped to popularize the smoking of cigarettes. Soldiers in the trenches smoked to relieve stress, and so did many civilians.

The link between the smoking of cigarettes and lung cancer began to be suspected by clinicians in the 1930s when they noted the increase of this "unusual" disease. Publications began to appear and about 2 decades later the role of smoking as causative agent had been firmly established. A case control study was published in 1940 in Germany and its author flatly stated that "the extraordinary rise in tobacco use was the single most important

cause of the rising incidence of lung cancer" (Müller, 1940). At this time, lung cancer had become the second most frequent cause of cancer death, stomach cancer being the first. In 1943, the German Institute for Tobacco Hazards Research disclosed a study which found that among 109 lung cancer cases only 3 were nonsmokers, a proportion much lower than in the control group. In the 1950s Doll and Hill in England and Cuyler Hammond and Ernest Wynder in the U.S. provided further evidence for a causal association between smoking and lung cancer. Yet, it took a long time until the truth was fully accepted. Smokers, including many physicians, who enjoyed cigarettes could or would not want to imagine or refused to believe that the habit (addiction would be more appropriate) was detrimental to their health. In the following decades, smoking continued to be "enjoyed" by hundreds of thousands until, after the first report of the Surgeon General in 1964, public awareness woke up and smoking became recognized as the hazard it is.

There was, however, one lung cancer where it had been obvious for a long time that it might be caused by an external agent. As early as 1500, attention was called to this particular condition. In two regions of Germany and Czechoslovakia,

Schneeberg and Joachimsthal, there were productive mines, yielding first silver, later nickel, cobalt, bismuth, and arsenic. The word "dollar" actually stems from the word "Thaler;" coins minted from the pure silver of Joachimsthal were called "Joachimsthaler" (i.e., originating from Joachimsthal) or, abbreviated, "Thaler." The miners working these mines developed almost invariably a deadly disease, called "Bergkrankheit" (mountain sickness). Between 1876 and 1938, 60 to 80% of all miners died from the disease which, on average, lasted 25 years. Certain regions of the mines were known as "death pits," where all workers got sick. As a result, lung cancer in miners was recognized as an occupational disease—and the miners therefore entitled for compensation—in 1926 in Germany and in 1932 in Czechoslovakia. While it was thought that chemical constituents of the ore that was produced, most notably arsenic, might be involved in the etiology of these lung cancers, it was early on suspected that "radium emanation" was the main culprit. Measurements published in 1924 in a German physics journal confirmed that the air in the mines contained high concentrations of radon gas, the highest more than 18,000 picocuires per liter.

DESCRIPTIVE EPIDEMIOLOGY

At the end of the 20th century, lung cancer had become one of the world's leading causes of preventable death. It was a rare disease at the start of that century, but exposures to new etiologic agents and an increasing lifespan combined to make lung cancer a scourge of the 20th century. While tobacco had been widely used throughout the world for centuries, the present pandemic of lung cancer followed the introduction of manufactured cigarettes with addictive properties, which resulted in a new pattern of sustained exposure of the lung to inhaled carcinogens.

There is a great variation in the prevalence of lung cancer in different geographical areas. Nearly 70% of all the new cases of lung cancer in the world occur in the developed countries. USA, Canada, New Zealand (Maori population) and Europe have the highest incidence (>50 per 105 population) followed by China, Ireland, Malta, Spain, Australia, and New Zeland (non - Maori population) with a moderate incidence (35-50 per 105 population) and low incidence (<35 per 105 population) countries include Utah (USA), Latin America, most Asian countries, lceland, Norway and Sweden. This is the most frequent tumour in males, and 2nd or 3rd

most common in females. In the US alone, there were about 1,64,100 new cases in 2000, of which 70,000 were in the metastatic stage (stage IV) and another 70,000 were locally advanced (stage IIIA and IIIB disease). In the European Union, the crude incidence of lung cancer is 52.2 cases per 105 per year and the death rate is 48.7 per 105 per year. For men, the rates are 79.3 and 78.3 and for women, 21.6 and 20.5 respectively per 105 per year. Non-small cell lung cancer accounts for about 80% of all lung cancer cases.

Some of the increases compared to that prior to 1950, may be due to improved diagnosis but changes more recently reflect an actual increase. In 1980, it was estimated to cause 15.8% of all new cancer cases in males varying between 4.5% in Africa to 23.3% in Europe. In females lung cancer is rare. However, the increase between 1975 and 1980 was 10.1% in males, but 16% in females. The situation was different in 1985. Ignoring the non-melanoma skin cancers, lung cancer was estimated to be the most common cancer in men in the world around 1985. It comprised 17.6% of all new cancers in men and 5.8% in women. In men there were about 667,000 new cases in 1985, and 219,000 in women. The

age adjusted mortality trends in 14 countries show that the increase is universal, at rates between one and five percent a year. Although the overall mortalities are less in females, marked increases have been seen in some countries, such as Canada, Denmark, and USA.

PATHOPHYSIOLOGY

Smoking

A single etiologic agent, cigarette smoking, is by far the leading cause of lung cancer, accounting for approximately 90% of lung cancer cases. Compared to never-smokers, smokers have about a 20-fold increase in lung cancer risk at present. Beedi smoking is more commonly seen among Indians of low socioeconomic groups and it is more carcinogenic than cigarette[14]. Passive smokers inhale a complex mixture of smoke that is now widely referred to as *environmental tobacco smoke* .

In the initial decades of the smoking-caused epidemic of lung cancer, squamous cell carcinoma was the most frequent type of lung cancer that was observed in the population among smokers, and small cell carcinoma was the next most frequent. In the late 1970s, the first evidence of a shift toward a predominance of adenocarcinoma was noted, and now adenocarcinoma of the lung is

the most common histologic type of lung cancer. In women, the Surveillance, Epidemiology, and End Results program data from 1973 to 1996 indicate that the incidence rates of squamous cell, small cell, and large cell carcinomas have at least reached a plateau while the rate for adenocarcinoma is still rising.

Occupation

Certain occupations carry a higher risk of lung cancer. The following occupational exposures are known to be associated with an increased risk: *(a) Asbestos:* insulation workers and shipyard workers are exposed to asbestos. There is some increase in the risk of lung cancer after 10 years of exposure and a substantial risk after 20 years of exposure. Concurrent smoking increases the risk to 90 fold; *(b) Arsenic :* smelter workers and vineyard workers are exposed to arsenic. The risk is dose related. Lung cancers have an upper lobe predominance and there may be multiple primaries; *(c) Nickel refinery workers :* squamous cell carcinoma is more common; *(d) Radiation (Uranium mining):* oat cell carcinoma is more common; *(e) Haematite mining:* due to radon exposure; *(f) Hard rock mining; (g) Chromium exposure in ore mining and pigment manufacturing*: squamous cell variety is most common; *(h) Chloromethyl exposure in workers in industries:* oat cell carcinoma

is most common; *(i) Ethers and mustard gas:* squamous and undifferentiated carcinomas are most common; *(j) Soot, tars exposure in coke oven* workers and (k) Oils and coke exposure in Gas house workers, roofers and rubber workers.

Air pollution

Air pollution increases the risk of lung cancer. Genetic susceptibility to lung cancer has long been postulated. Environmental agents, even cigarette smoking, cause lung cancer in only a minority of exposed persons, leading to the hypothesis that susceptibility is inherently determined. People who eat more vegetables are at lower risk of lung cancer than persons who consume fewer vegetables. The same may also apply to fruit consumption, but the evidence is less clear-cut.

Genetics

Recently, advanced molecular techniques have identified amplification of oncogenes and inactivation of tumor suppressor genes in NSCLC and SCLC. The most important abnormalities detected in NSCLC are mutations involving the *ras* family of oncogenes. The *ras* oncogene family has 3 members: H-*ras,* K-*ras,* and N-*ras*. These genes encode a protein on the inner surface of

the cell membrane with GTPase activity and may be involved in signal transduction. To what extent these changes are causal events in the development of SCLC is not clearly known and remains an area of active research. Amplification of the *myc* family of oncogenes is the most common molecular abnormality identified in SCLC cell lines, xenografts in nude mice, and fresh tumor specimens. This change, however, is not identified in all SCLC tumors. Though it is not an initial event in the pathogenesis of SCLC, C-myc and N-myc amplification carry a poor prognosis. The retinoblastoma (*RB*) tumor suppressor gene, is on chromosome 13 (13q14), and a high percentage of SCLCs (as many as 60%) do not express *RB* messenger ribonucleic acid (mRNA). This high frequency of inactivation of a tumor suppressor gene suggests that this may be an important step in the molecular pathogenesis of SCLC. The most common molecular abnormality, however, is deletion of part of chromosome 3 (3p14). Mutations of the *p53* tumor suppressor gene are found commonly in both SCLC and NSCLC, but their precise role in pathogenesis is not clear. Tobacco smoking and radon exposure are associated with *p53* gene mutations .

Classification

Lung carcinoma is generally classified as small cell (SCLC) and non–small cell (NSCLC). Non–small cell lung cancer accounts for approximately 75% of all lung cancers. NSCLC is divided further into adenocarcinoma, squamous cell carcinoma, and large cell carcinoma. All share similar treatment approaches and prognoses but have distinct histologic and clinical characteristics. Small cell carcinomas arise in peribronchial locations and infiltrate the bronchial submucosa. It exhibits aggressive behaviour with widespread metastases occuring early in the course of the disease, with common spread to mediastinal lymph nodes, liver, bones, adrenal glands, and brain. In addition, production of a variety of peptide hormones leads to a wide range of paraneoplastic syndromes. The most common paraneoplastic syndromes are the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and the syndrome of ectopic adrenocorticotropic hormone (ACTH) production. In addition, autoimmune phenomena may lead to various neurological syndromes.

CLINICAL FEATURES

Symptoms, signs relating to the lung cancer can be classified as follows: (1) those related to the primary lesion, (2) those related to intrathoracic spread, (3) those related to distant metastasis, and (4) those related to paraneoplastic syndromes[8].

(1)Signs and symptoms related to lung cancer:

Cough is the most common presenting symptom in lung cancer. Many lung cancers occur in central airways and may lead to postobstructive pneumonia, or cause lymph node enlargement that may lead to cough. Failure of acute exacerbations of COPD to clear should raise the suspicion of a neoplasm.

Dyspnoea is usually associated with increasing cough and sputum. If the tumor is occluding a main airway, it can cause breathlessness, which may be associated with a unilateral wheeze.

Hemoptysis is rarely severe and usually only consists of blood streaking of the sputum. In patients > 40 years old with COPD and a smoking history presenting with hemoptysis, there should still be a high index of suspicion for lung cancer.

Chest discomfort is often of an ill-defined nature, intermittent and aching in quality. Definite pleuritic pain may occur as a result of direct spread of the tumor to the pleural surface.

(2)Signs and symptoms of intrathoracic spread:

Recurrent laryngeal nerve palsy is more common in left-sided tumors because of the circuitous route of the left recurrent laryngeal nerve around the aortic arch, and causes hoarseness. It is associated with poor expectoration with coughing, and an increased risk of aspiration.

Phrenic nerve dysfunction may be noted on the chest radiograph by the presence of an elevated hemidiaphragm, or can present with breathlessness in patients already compromised by lung disease.

Pancoast tumor is also called superior sulcus tumor, and arises posteriorly in the apex of an upper lobe near the brachial plexus, commonly infiltrating the eighth cervical and first and second thoracic nerve roots. This causes pain, cutaneous temperature change, and muscle wasting along the relevant nerve

root. There is often a delay of many months before the true diagnosis is revealed.

Horner's syndrome is due to involvement of the sympathetic chain and stellate ganglion causing unilateral enophthalmos, ptosis, small pupil, and ipsilateral lack of facial sweating.

The chest pain is usually dull, tends to be persistent, poorly localized, and unrelated to breathing or coughing. Retrosternal pain may be due to massive hilar and mediastinal nodal involvement. When chest pain is particularly severe and localized, it is usually related to either direct invasion of the pleura or chest wall by the primary tumor, or due to a rib metastasis. Tenderness may be elicited at the site of rib involvement and, rarely, a soft-tissue mass can be palpated.

Pleural effusion, which may result in dyspnea, is generally caused by direct pleural extension, but may also be secondary to mediastinal node involvement and lymphatic obstruction. Signs of a pleural effusion include dullness to percussion and decreased breath sounds.

Lung cancer accounts for 46 to 75% of all cases of ***SVC obstruction*** *(SVCO);* the most common histologic subtype associated with SVCO is small cell carcinoma and is due to direct invasion by the primary tumor, or from enlarged right paratracheal metastatic lymph nodes. The patient will complain of facial swelling, including the neck and eyelids, with dilated veins visible over the upper torso, shoulders, and arms. There may also be headache, dizziness (particularly on bending forwards), drowsiness, blurring of vision, cough, and dysphagia.

Metastases to the heart and pericardium usually occur by direct lymphatic spread. At autopsy, cardiac involvement occurs in approximately 15% of cases, and a small number will have tamponade. In primary lung cancer, the pericardium is the most common site of cardiac involvement.

Enlargement of hilar and mediastinal nodes is usually due to metastatic spread and seldom causes symptoms unless it is massive, when it can compress the esophagus and cause ***dysphagia***.

(3)Symptoms and signs of extrathoracic metastasis:

Approximately one third of patients present with symptoms as a result of distant metastases. The most common sites of distant

metastasis from lung cancer are the bones; liver, adrenal glands, and intraabdominal lymph nodes; brain and spinal cord; and lymph nodes and skin.

The axial skeleton and proximal long ***bones*** are most commonly involvedand the primary symptom resulting from bone involvement is pain.

Liver metastases occur commonly with lung cancer. However, liver function test results are seldom abnormal until the metastases are numerous and large. Hepatic metastases most commonly produce symptoms of weakness and weight loss. Hepatic metastases carry a very poor prognosis.

Adrenal lesions and para-aortic lymph node metastases may occur and are most commonly seen with small cell lung cancers; in the latter cell type, they are often discovered during staging. Intracranial metastases occur in 10% of patients at presentation.

Brain metastasis may produce headache, nausea and vomiting, focal neurologic symptoms or signs, seizures, confusion, and personality changes. The lung is the primary site of

approximately 70% of cancers that initially present with symptomatic brain metastases.

The most common site of ***palpable lymphadenopathy*** is the *supraclavicular* fossa, which can be involved in 15 to 20% of cases during the course of the disease. Identifying an enlarged lymph node or subcutaneous nodule due to metastatic lung cancer is extremely helpful in facilitating both diagnosis and staging. Fine needle aspiration can be performed quickly at the bedside or as an outpatient with little morbidity and with a high sensitivity.

(4)Paraneoplastic syndromes:

Paraneoplastic syndromes are a group of clinical disorders associated with malignant diseases that are not directly related to the physical effects of primary or metastatic tumor. The exact mechanism by which paraneoplastic syndromes occur is not fully understood. Paraneoplastic syndromes occur in at least 10% of patients with bronchogenic carcinoma.

Hypercalcemia is frequently secondary to bony metastases. It can, however, be due to production of a parathyroid hormone-related peptide. It is most common with squamous cell carcinoma;

approximately 15% of patients acquire hypercalcemia before death. Symptoms include nausea, vomiting, abdominal pain, constipation, polyuria, thirst, dehydration, confusion, and irritability.

SIADH Syndrome of inappropriate Anti Diuretic hormone is mainly associated with small cell lung cancer, although other malignant tumors of the lung may rarely be associated with this syndrome. Manifestations of SIADH include confusion, unexplained seizures, decreased level of consciousness, and coma. Biochemically, the syndrome is defined as low serum sodium, a dilute plasma osmolality with a higher, or "inappropriate" urine osmolality, in the presence of continued urinary sodium excretion. The syndrome resolves promptly (< 3 weeks) with the initiation of combination cytotoxic chemotherapy in 80% of patients with

small cell lung cancer, but commonly recurs with tumor progression. ***Cushing syndrome***-Adrenocorticotrophic hormone (ACTH) is the most commonly produced ectopic hormone in lung cancer. But Cushing syndrome is rarely seen. Signs and symptoms of Cushing syndrome develop in only 1 to 5% of patients with small cell lung cancer. The most common features associated with this condition are clinical symptoms of weakness, muscle

wasting, drowsiness, confusion and psychosis, dependent edema, hypokalemic alkalosis, and hyperglycemia.

Digital Clubbing and Hypertrophic Osteoarthropathy may be associated with any cell type of lung cancer, although they are most frequently associated with squamous and adenocarcinoma and least frequently associated with small cell lung carcinoma. The exact mechanism for clubbing and hypertrophic osteoarthropathy is unknown, although suggestions include neurogenic, hormonal, and vascular mechanisms. Clubbing is much more common than hypertrophic osteoarthropathy. Hypertrophic osteoarthropathy is seen in < 5% of patients with non small cell lung cancer and is characterized by a painful symmetrical arthropathy (usually of the ankles, wrists, and knees) and periosteal new bone formation on the distal long bones of the limbs.

A variety of poorly understood ***neurologic syndromes*** may occur in lung cancer, and affect 4 to 5% of patients. Small cell carcinoma is the most common type of lung cancer associated with paraneoplastic autoimmune neurologic syndromes.The syndromes involved include Lambert-Eaton myasthenic syndrome, peripheral neuropathy, cortical cerebellar degeneration, and several other

CNS syndromes. Some of these syndromes may be identified by the presence of antibodies either in the serum or the cerebrospinal fluid.

DIAGNOSIS

Once the signs and symptoms suggest the possibility of lung cancer,the next step is to proceed with a x-ray chest and computed tomography(CT) of the chest. Imaging plays an integral role in diagnosing, staging, and following patients with lung cancer. Most lung tumors are detected on chest radiographs, but unfortunately, the majority of patients have advanced stage disease at presentation. There is a wide spectrum of radiologic manifestations of lung cancer, and recognition of these findings is essential for patient management.The radiographic findings and clinical presentation usually allow a presumptive differentiation between small cell lung cancer (SCLC) and non-SCLC (NSCLC). Massive lymphadenopathy and direct mediastinal invasion are well-recognized phenomena in *SCLC*. A mass in, or adjacent to, the hilum is a particular characteristic of SCLC and is seen in about 78% of cases. Among NSCLC, ***adenocarcinoma*** typically presents as a small (often < 4 cm), peripheral, round or oval, smoothly marginated, solitary pulmonary nodule.

Bronchioloalveolar cell carcinoma (BAC) is a peculiar subtype of adenocarcinoma that may present with solitary or multiple lesions. Multifocal BAC may present as follows: (1) multiple well-defined nodular opacities of varying size, involving one or both lungs (15%) ; (2) focal, poorly defined opacities or multiple scattered opacities that may coalesce into lobar and rarely complete lung opacification, resembling pneumonia (10%); or (3) reticulonodular opacities resembling interstitial lung disease.

Squamous cell carcinoma usually range in size from 1 to 10 cm. They are typically found in the central bronchi, although one third occur beyond the segmental bronchi. Endobronchial neoplasm may result in postobstructive pneumonia and/or atelectasis in up to 50% of cases, and the underlying mass may be observed. Squamous cell carcinoma cavitates in 10 to 20% of cases, particularly in large peripheral lesions (30%). Cavity walls are usually thick and irregular, ranging in size from 0.5 to 3 cm and here it has to be differentiated from lung abscess. Rarely, extensive necrosis may result in a thin-walled cavity. Squamous cell carcinoma is the most common type to prove Pancoast or superior sulcus tumors.Asymmetry of > 8 mm in apical pleural thickening may be

an important finding, especially when associated with chest wall pain, brachial or laryngeal nerve paralysis, or bone destruction.

The majority of ***large cell carcinoma*** present as a large (average size > 7 cm), peripheral mass, with poorly defined margins. Cavitation and calcification are uncommon (6%). Hilar and mediastinal adenopathy are seen in 30% and 10% of cases, respectively. Rapid growth with early lymphatic and hematogenous metastases occurs frequently.

In asymptomatic patients, ***a solitary pulmonary nodule*** may be an incidental finding in chest x-ray. A solitary pulmonary nodule (SPN) is radiologically defined as an intraparenchymal lung lesion that is < 3 cm in diameter and is not associated with atelectasis or adenopathy[23]. Lung lesions > 3 cm in size are defined as lung masses. One of 500 chest radiographs (CXRs) demonstrates a lung nodule. Ninety percent of these are incidental radiologic findings, found unexpectedly in radiographs obtained for unrelated diagnostic workups. The tragedy of lung cancer is directly associated with its delayed presentation. Signs and symptoms are rarely present until the malignancy has become advanced and possibly unresectable. Patients with the best prognosis are those

found to have stage IA (T1N0M0) disease. Therefore, a timely and accurate diagnosis of the etiology of an SPN is essential to providing the patient with malignancy a potential for cancer cure. The occult nature of a lung nodule with its few symptoms and inability to be detected on physical examination does not lend itself to the sense of immediacy prompted by the discovery of other potential malignancies, such as a breast mass. The differential diagnosis of an SPN includes neoplastic, infectious,inflammatory, vascular, traumatic, and congenital lesions. Other benign etiologies for SPNs are rheumatoid nodules, intrapulmonary lymph nodes, plasma cell granulomas, and sarcoidosis. Although most SPNs are benign,primary malignancy may be found in approximately 35% of SPNs, and solitary metastases can account for another 23%. Traditionally the presence of "benign" calcification or the absence of growth over a 2-year time period has been believed to be reliable indicators of benign disease. Benign calcification refers to central, diffuse, laminar, or popcorn patterns. Other types of calcifications such as eccentric or stippled calcifications are radiologically "indeterminant" and are seen in both benign and malignant lesions. CXRs may also falsely suggest that calcium is present, leading the clinician and patient to have false confidence

that the nodule is benign. In a recent series by Berger et al,7% of nodules that were believed to be "definitely calcified" by the CXR lacked calcium on the CT scan. Benign lesions typically have a doubling time of either < 1 month or > 16 months. Malignant nodules have a doubling time from anywhere from 40 to 360 days. The morphology of a nodule that is spherical with rounded edges is associated with benign disease. Spiral CT with IV contrast enhancement is the imaging modality of choice for the SPN and should be obtained on all newly diagnosed SPNs.

Computed tomography is now established as the best non-invasive method for staging the lung cancer by assessing the chest wall invasion, pleural invasion, mediastinal invasion and mediastinal lymph node

involvement. It is also useful in detecting early stage of lung cancer where x-ray appears normal. Radiologic studies used in conjunction with the International System for Staging Lung Cancer include chest radiographs, CT, and occasionally MRI. The appropriate role of imaging in management still requires definition, but the major indication is to accurately differentiate stage I to IIIA (potentially resectable) from stage IIIB to IV (nonresectable)

cancer. Current imaging for bronchogenic carcinoma makes use of plain chest radiographs, CT, MRI, and nuclear medicine.

NSCLC: Radiologic assessment of the *size of primary* lesions is usually done using plain chest radiographs and, less commonly, CT or MRI. CT and occasionally MRI are used to evaluate the *hilar and mediastinal lymph nodes*. Size, unfortunately a nonspecific criterion, is the only criterion used in attempting to distinguish normal from abnormal lymph nodes (short axis > 1 cm is considered abnormal). Lymph node morphology and MRI signal characteristics are not useful in predicting lymph node metastases.Although CT and MRI are very accurate in demonstrating enlarged lymph nodes, the cause of enlargement may be reactive hyperplasia, not metastasis, particularly if there is a postobstructive pneumonia.The accuracy of CT and MRI for detecting metastatic hilar (N1) disease is only 62 to 68% and 68 to 74%, respectively[20]. This low accuracy of radiographic staging of N1 metastatic disease, in most cases, does not prevent surgical resection unless the patient is a poor surgical candidate. Common sites of *metastases* are lymph nodes as described above; brain and CNS, bone, and adrenal gland metastases and metastases to the contralateral lung are considered M1 disease. Initiating a

radiologic investigation for metastatic disease is often based on the clinical history, physical examination, and blood indexes (CBC count, alkaline phosphatase, liver function tests).Routine radiologic evaluation for occult metastases in the absence of clinical or laboratory findings is not clearly indicated. Isolated CNS metastases are rare in patients with NSCLC and are generally associated with an abnormal neurologic examination. Asymptomatic brain metastases occur in 2.7 to 9.6% of patients usually with large cell carcinomas and adenocarcinoma. The use of routine CT or MRI of the CNS in asymptomatic patients with NSCLC is controversial. Patients with bone metastases are usually symptomatic (pain) or have suggestive laboratory abnormalities (*eg*, elevated alkaline phosphatase). Bone radiographs, radionuclide bone scanning with 99 technetium-methylene diphosphonate or MRI are useful modalities for further investigation. Occult skeletal metastases are rarely (up to 4%) detected by radionuclide 99 technetium-methylene diphosphonate studies, but there is a high false-positive rate (approximately 40%).Thus, routine radionuclide skeletal imaging should not be performed in NSCLC. Adrenal metastases do not produce reliable clinical and laboratory findings; thus, upper abdominal imaging is

routinely performed, especially as part of thoracic CT staging. Incidental nonfunctioning cortical adenomas occur in 3 to 5% of the population, and approximately 10% of patients with NSCLC will have an adrenal mass at CT.In the absence of other known extrathoracic metastases, an adrenal mass is more likely benign. Attenuation values ≤10 are virtually pathognomonic benign adrenal enlargement. CT and MRI are similar in detecting hepatic metastases, although isolated liver metastases are extremely uncommon and routine liver imaging is not usually suggested.

SCLC: SCLC has been divided into two stages i.e., limited and extensive disease. Limited disease is defined as tumor within a single radiotherapy port (tumor confined to the thorax).

Extensive disease includes distant metastases and noncontiguous metastases to the contralateral lung. Long-term survival occurs primarily with limited disease and is rare with extensive disease. Extensive disease is present at presentation in 60 to 80% of patients with SCLC. Metastases commonly occur in the liver (22 to 28%), bone (30 to 38%), bone marrow (17 to 25%), brain (8 to 15%), and retroperitoneal lymph nodes (11%). Abdominal CT or ultrasound should be done routinely in the

staging evaluation of SCLC. MRI may be more sensitive than contrast-enhanced CT in detecting hepatic metastases, and similar to CT in evaluation of adrenal metastases. Isolated bone and bone marrow metastases are uncommon and are usually associated with involvement of other organs.

Consequently, if there are extrathoracic metastases, further evaluation should include a radionuclide bone scan and bone marrow aspiration. MRI may be more sensitive than bone scintigraphy in detecting small and rapidly growing metastases with marrow infiltration. CNS metastases are common at presentation and as a site of future disease. Routine CT or MRI evaluation of the CNS is recommended, as approximately 5% of patients with cerebral metastases are asymptomatic. In NSCLC, 2.7 to 9.6% are symptomatic and use of imaging is controversial. Detection and treatment with aggressive chemotherapy and radiotherapy can decrease morbidity and improve prognosis if the brain is the only site of extrathoracic disease.

After the radiological diagnosis and staging, the next step is to achieve a *definitive diagnosis*.

Sputum cytology is the least invasive means of obtaining a diagnosis in a patient who is suspected of having lung cancer. The diagnostic accuracy of sputum cytology is dependent on rigorous specimen sampling (at least three specimens) and preservation techniques, as well as on the location and size of the tumor (*ie*, central vs peripheral). Unfortunately, many institutions do not have an established program for sputum collection and processing, and therefore sputum analysis has a much lower sensitivity than that indicated in the data presented here (which come from institutions with well-established sputum analysis programs). Sputum cytology is particularly useful in patients who present with centrally located tumors (*ie*, SCLC or squamous call carcinoma) and in those who present with hemoptysis.

Flexible bronchoscopy with its attendant procedures is a valuable diagnostic procedure in the workup of a patient who is suspected of having lung cancer. The decision about whether to perform a diagnostic bronchoscopy for a lesion that is suspicious for lung cancer largely depends on the location of the lesion (*ie*, central vs peripheral). In a patient with a central lesion, bronchoscopy is the most sensitive way to confirm a diagnosis of cancer[19]. Central lesions can present as an exophytic endobronchial

mass, submucosal spread, or a peribronchial tumor causing extrinsic compression. Direct forceps biopsy of visible central lesions is the technique used most frequently. At least three forceps biopsies of the visible lesion are recommended. The sensitivity from washings and brushings is somewhat lower, but these tests often are combined with forceps biopsies. The addition of *Transbronchial Needle Aspiration* to obtain cytology or histology samples when there is submucosal tumor spread or peribronchial tumor causing extrinsic compression increases the sensitivity of bronchoscopy. Peripheral lesions are defined in most studies as lesions that are not visible in the main or lobar airways, and thus it is not surprising that the sensitivity of flexible bronchoscopy for diagnosing peripheral lung cancers is lower than that for central lesions. A few points must be made in order to interpret the results of bronchoscopy in the diagnosis of peripheral lung cancers. First, most of the studies used fluoroscopy routinely for peripheral lesions, which increases the reported sensitivity of bronchoscopy. In a patient with a small (*ie*, < 2 cm) peripheral lesion, the sensitivity of bronchoscopy is low. The false-negative (FN) rate for bronchoscopy results has not yet been defined. Most clinicians would pursue the diagnosis further in the case of a

nondiagnostic bronchoscopy of a visible endobronchial abnormality. The FN rate can be estimated to be fairly high in the case of peripheral lesions, especially smaller ones, because of the relative low sensitivity in this setting. Bronchoscopy has an important role in the diagnosis of benign conditions, but the chance of finding a benign condition in a patient who is clinically suspected of having lung cancer is only 1%.

Percutaneous transthoracic needle aspiration (TTNA) biopsy is a commonly used diagnostic procedure when a lung tumor is diagnosed. It is generally regarded as a safe procedure with limited morbidity and extremely rare mortality. The most common complication is a pneumothorax that will occasionally require a chest tube. Since TTNA is highly sensitive for diagnosing malignancy,1 a diagnosis of malignancy obtained by TTNA can be regarded as having a high predictive value and the patient should be treated accordingly. In some cases, however, the results from TTNA can be nonspecific. Since many patients are not surgical candidates, TTNA is a necessary procedure to obtain a tissue diagnosis before starting radiation therapy or chemotherapy. In patients with a solitary pulmonary nodule, one could calculate the probability of malignancy as described by Cummings et al.18

Cummings and coauthors18 used Bayes' theorem to develop a simple method for combining individual clinical features of patients with solitary pulmonary nodules (specifically the size of the nodule, the patient's age and smoking history, and the clinical setting in which the nodule was discovered) into an overall estimate of the probability that the nodule is malignant. For patients who have a low probability of malignancy, TTNA is probably not the most ideal diagnostic method since it has a low sensitivity for benign tumors. Surgery would not be the best choice either since there would be an increased likelihood of unnecessarily operating on a benign lesion. Instead, core biopsy could be utilized in these patients since it has been shown to be very effective at diagnosing benign lesions. This technique may be more likely to produce a pneumothorax, but it will avoid the need for a surgical procedure. Conversely, if a patient has a pulmonary tumor with a high likelihood of being malignant, and that patient is a surgical candidate, then immediate surgery is most likely to be the optimum treatment method. If this hypothetical patient underwent TTNA and this resulted in a nonspecific diagnosis, then the patient would prob-ably be referred for open surgical biopsy. If this same patient underwent TTNA and was diagnosed as having a

malignancy, then the eventual treatment would still be surgical resection. For patients such as this, TTNA adds risk and cost but does not change the treatment plan for the patient. TTNA is most useful for diagnosing pulmonary nodules that are highly likely to be malignant in patients who are not candidates for other surgical procedures.

Lymph node biopsy: Tissue sampling from obvious metastatic sites is yet another way by which the diagnosis of lung cancer can be established.

If a diagnosis is not well established y any of the above procedures a thoracotomy may be necessary, however this decision is not taken lightly and the general guideline is the procedure undertaken only when a potential cure from lung cancer can be anticipated.

STAGING

The stages represent the nature and extent of spread of a neoplasm and, thus, the therapeutic options and prognosis in individual patients. Stages also provide a standard by which various therapies can be compared. A combination of clinical,

laboratory, radiologic, and pathologic investigations are used to stage various neoplasms.

Different staging systems have been developed. The most widely used scheme for staging NSCLC is the TNM classification. The UICC and the AJCC initially introduced this system in 1972 with staging and end-results reporting. This scheme has been modified and refined over the years. The TNM classification in the publications of the UICC and the AJCC are identical. They were formulated together but appear in separate books—namely, the *UICC TNM Classification of Malignant Tumors* and the *AJCC Cancer Staging Manual.* Both are in their sixth editions and were published in 2002.

TNM is a dual system with a pretreatment Clinical classification (cTNM or TNM) and a postsurgical histopathologic Pathological classification (pTNM[6]). Both classifications are retained unaltered in the patient's record. The former is used for the choice of treatment; the latter is used for the estimation of prognosis and the possible selection of adjuvant therapy.

The TNM staging system takes into account the degree of spread of the primary tumor, represented by T; the extent of

regional lymph node involvement, represented by N; and the presence or absence of distant metastases, represented by M. The TNM system is used for all lung carcinomas except SCLCs, which are staged separately.

In the TNM systems, 4 stages are further subdivided into I-III and A or B subtypes. These stages have important therapeutic and prognostic implications. The stages are as follows.

Primary tumor

Tis - Carcinoma in situ

TX - Positive malignant cytologic findings, no lesion observed

T1 - Diameter of 3 cm or smaller and surrounded by lung or visceral pleura or endobronchial tumor distal to the lobar bronchus

T2 -Diameter greater than 3 cm; extension to the visceral pleura, atelectasis, or obstructive pneumopathy involving less than 1 lung; lobar endobronchial tumor; or tumor of a main bronchus more than 2 cm from the carina

T3 - Tumor at the apex ; total atelectasis of 1 lung; endobronchial tumor of main bronchus within 2 cm of the carina but not invading it; or tumor of any size with direct extension to the adjacent

structures such as the chest wall mediastinal pleura, diaphragm, pericardium parietal layer, or mediastinal fat of the phrenic nerve

T4 - Invasion of the mediastinal organs, including the esophagus trachea, carina, great vessels, and/or heart; obstruction of the superior vena cava; involvement of a vertebral body; recurrent nerve involvement; malignant pleural or pericardial effusion; or satellite pulmonary nodules within the same lobe as the primary tumor.

Regional lymph node involvement

N0 - No lymph nodes involved

N1 - Ipsilateral bronchopulmonary or hilar nodes involved

N2 - Ipsilateral mediastinal nodes or ligament involved

Upper paratracheal lower paratracheal nodes

Pretracheal and retrotracheal nodes

Aortic and aortic window nodes

Para-aortic nodes

Para-esophageal nodes

Pulmonary ligament

Subcarinal nodes

N3 - contralateral mediastinal or hilar nodes involved or any scalene or supraclavicular nodes involved.

Metastatic involvement

M0 - No metastases

M1 - Metastases present

Stage groupings are as follows:

IA - T1N0M0

IB - T2N0M0

IIA - T1N1M0

IIB - T2N1M0 or T3N0M0

IIIA - T1-3N2M0 or T3N1M0

IIIB - Any T4 or any N3M0

IV - Any M1

Staging of SCLC

SCLC has usually metastasized by the time of presentation. Extensive disease is present in more than 60% of patients. Surgery is an option in fewer than 5% of these patients, who have a small primary and no evidence of spread. Systemic chemotherapy is the main treatment, with response rates of 70% but cure rates of less than 5%.

The distinction between limited and extensive disease is an important staging issue. The TNM system may be applied, but it is not directly relevant to management decisions. The objectives of

staging in SCLC are to identify localized disease, for which radiation therapy may be suitable, and to quantify the extent of the disease before therapy.

Localized disease is defined as disease confined to 1 hemithorax that includes ipsilateral, contralateral, and/or supraclavicular nodes. Investigations include chest radiography; CT of the thorax, liver, and adrenal glands; cranial CT; bone scintigraphy; and bone marrow aspiration. The disseminated nature of SCLC makes whole-body survey techniques suitable for its evaluation. 99mTc-labeled monoclonal antibody fragment NR-LU-10 is used to detect an antigen present in most small cell cancers.

Pulmonary Function Testing

Many patients with NSCLC have moderate to severe chronic lung disease that increases the risk of perioperative complications as well as long-term pulmonary insufficiency following lung resection. All patients considered for surgery require spirometry. In the absence of other comorbidities, patients with good lung function (preoperative FEV_1 > 2 L) are at low risk for complications from lobectomy or pneumonectomy.

Screening

The optimal screening of smokers for lung cancer is unclear. Both chest radiographs and analysis of expectorated sputum is ineffective. Recent studies have tried to assess the role of chest CT scans. A study by Bach et al showed that CT screening of populations increases the diagnosis of lung cancer but does not reduce the risk of advanced lung cancer or reduce mortality. The investigators of the International Early Lung Cancer Action Program, however, suggested that there is a role for screening with chest CT. These studies highlight how complex and confusing the analysis of screening for lung cancer can be. At the present time, there are inadequate data to recommend the use of chest CT to screen smokers for lung cancer. The National Lung Screening Trial is an ongoing large, multicenter study that should answer the question definitively.

MANAGEMENT OF LUNG CANCER

Non–small cell carcinoma

Cure of NSCLC is unlikely without resection. Clinical features that preclude complete resection include extrathoracic metastases or a malignant pleural effusion; or tumor involving the heart, pericardium, great vessels, esophagus, recurrent laryngeal or phrenic nerves, trachea, main carina, or contralateral mediastinal lymph nodes.

Accordingly, stage I and stage II patients are treated with surgical resection where possible, and stage II are additionally recommended to receive adjuvant chemotherapy[46].

Stage IIIA patients have poor outcomes when treated with resection alone. They should be referred to multimodality protocols, including chemotherapy and radiotherapy.

Stage IIIB patients treated with combined chemotherapy and radiation therapy have improved survival. Selected stage IIIB patients taken to resection following multimodality therapy have shown long-term survival and may be cured.

Stage IV patients are treated with symptom-based palliative therapy, which may include outpatient chemotherapy. Neoadjuvant

chemotherapy consists of giving antineoplastic drugs in advance of surgery or radiation therapy.

There is no consensus on the impact of neoadjuvant therapy on survival in stage I and stage II NSCLC. Such therapy is not recommended outside of ongoing clinical trials. Neoadjuvant therapy is more widely used in selected patients with stage IIIA or stage IIIB disease. Some studies suggest a survival advantage. This remains an area of active research. Adjuvant chemotherapy consists of administering antineoplastic drugs following surgery or radiation therapy. Adjuvant chemotherapy with alkylating agents such as cyclophosphamide increases mortality. In stage I and N0 stage II disease, patients treated with multidrug platinum-based chemotherapy show a trend toward improved survival—on the order of 3 months at 5 years. Toxicity may be significant, however, and such therapy is not routine. Newer antineoplastic agents with less toxicity are in clinical trials in these patients, and gene expression profiling has shown promise in defining a subset of patients with stage IA disease who may benefit. In patients with stage IIIA disease and node positive stage II disease, the data are conflicting whether chemotherapy following surgery improves survival. Patients with locally advanced disease (stages IIIA and

IIIB) who are not surgical candidates have improved survival when treated with combination chemotherapy and radiation therapy compared with no therapy or radiation alone.

Small cell carcinoma

Response rates of SCLC to cisplatin and etoposide are excellent: 80–100% response in limited-stage disease (50–70% complete response), and 60–80% response in extensive stage disease (15–40% complete response). However, remissions tend to be short-lived with a median duration of 6–8 months. Once the disease has recurred, median survival is 3–4 months. Overall 2-year survival is 20% in limited-stage disease and 5% in extensive-stage disease. Thoracic radiation therapy improves survival in patients with limited SCLC but not those with extensive disease. Whole brain radiation therapy decreases the incidence of central nervous system disease but does not affect survival. Its effect on symptoms is controversial.

Occasionally, a patient may have a peripheral nodule resected that turns out to be SCLC. Five-year survival following resection of the equivalent of stage I and stage II SCLC is higher than in patients treated with chemotherapy.

Palliative therapy

Photoresection with the Nd:YAG laser is sometimes performed on central tumors to relieve endobronchial obstruction, improve dyspnea, and control hemoptysis. External beam radiation therapy is also used to control dyspnea and hemoptysis, pain from bony metastases, obstruction from superior vena cava syndrome, and symptomatic brain metastases. Resection of *solitary* brain metastases does not affect survival but may improve quality of life when combined with radiation therapy. Intraluminal radiation (brachytherapy) is an alternative approach to endobronchial disease. Pain syndromes are very common in advanced disease. As patients approach the end of life, meticulous efforts at pain control are essential. Consultation with or referral to a palliative care specialist is recommended in advanced disease to aid in symptom management and to facilitate referrals to hospice programs.

Prognosis

The overall 5-year survival rate for lung cancer is 15%. Predictors of survival are the type of tumor (SCLC versus NSCLC), the stage of the tumor, and the patient's performance status, including weight loss in the past 6 months. These are independent predictors in both early and late stage disease. Most

data suggest that there is no difference among non–small cell carcinomas when adjusted for stage and performance status. However, squamous cell carcinoma may have a better prognosis than adenocarcinoma or large cell carcinoma at the same TNM stage.

Since lung cancer carries the highest mortality among all cancers, and no treatment carries a best prognosis, prevention of lung cancer through reduction of tobacco use is the primary stratergy that will result in marvelous reduction in lung cancer incidence.

MATERIALS AND METHODS

The study was conducted in Government Rajaji Hospital, Madurai. Lung cancer patients from various departments such as Medical oncology, General medicine, Thoracic medicine and Cardiothoracic surgery wards were proposed to collected for study. Ethical committee approval was obtained for the study.

TYPE OF STUDY – PROSPECTIVE STUDY

Inclusion criteria

All the patients admitted with clinical picture and Imaging suggestive of lung cancer, and later proved to be of primary lung malignancy were included in the study.

Exclusion criteria

Multiple mass lesion lung suggestive of secondaries but without evidence of primary lung mass.

Multiple mass lesion lung with hilar mass but without any evidence of growth by bonchoscopy.

Mass lesion lung suggestive of primary lung cancer but biopsy not confirmed.

The study was conducted during the period between July 2006 and June 2007. Totally fifty newly diagnosed lung cancer patients were collected for the study. The main inclusion criteria for the study were patients with histological evidence of lung cancer in those with mass lesion lung. Mass lesion lung patients in whom histological evidence could not be made because of the non consent by patients were excluded from the study.

Elaborative clinical data were collected from these patients. They include age, sex, presenting complaints cough, chest pain, dyspnoea, hemoptysis, hoarseness of voice, neural pain along ulnar border, facial puffiness, dysphagia, fever, bone pain, weakness, neck swelling, headache, seizures, focal neurological deicits, bony and skin swellings. Smoking history was collected that revealed whether they smoked cigarette, beedi or both, number of cigarettes/beedis smoked per day, number of years of smoking and h/o passive smoking. Past history of pulmonary tuberculosis and Family history of lung cancer and other cancers were also probed through. All patients were examined for clubbing, e/o lymphadenopathy, e/o paraneoplastic syndromes, e/o consolidation, collapse and pleural effusion. Also the patients were subjected for examination of hepatomegaly with nodules, cutaneous and bony

swellings, papilloedema, focal neurological deficits to identify metastasis.

All patients were taken x-ray chest postero anterior view. and they were looked for mass lesion lung and its location, mediastinal widening, pleural effusion, rib infiltraion, multiple lung secondaries. For confirmation and staging purpose they were all done a computed tomography of chest and noted for mass lesion lung and its location, mediastinal nodes, mediastinal invasion, pericardial effusion and invasion, pleural effusion, pleural invasion and nodules, chest wall infiltraion, multiple lung secondaries. For the same they were all subjected to ultrasonogram abdomen to study for abdominal secondaries.For those patients whom had symptoms and signs of central nervous system were subjected for computed tomography brain for proof of secondaries. Only two patients whom had bony symptoms did a bony scitigraphy done outside our hospital. They came here with those results and consulted us for the first time.

And only thirty patients consented for bronchoscopy and they were examined for bronchial growth, carina widening, tracheal invasion and vocal cord palsy by our thoracic physicians. They

also underwent biopsy of the growth and bronchoalveolar lavage was done in those patients and sent for histopathological study. In other patients, with their written consent, tissue for histopathological proof was attempted with CT guided trucut biopsy done by radiologist, supraclavicular node biopsy done by surgeons and the tissue was sent for the histopathological investigation.

Based on all above mentioned clinical features and investigation staging was done.

RESULTS

Out of 89 patients who were admitted for suspected lung cancer only fifty patients consented for imaging and procedures and they were included for study. The remaining 39 patients were excluded from the study because of 1) their denial for imaging due to financial constraints, 2)their denial for invasive procedures due to their illiteracy leading to fear, 3)their denial for prolonged hospital stay since some of the patients had come from a long distance for tertiary care.

AGE AND SEX

TABLE 1: SEX DISTRIBUTION

Sex	No: of patients(n) (Total-50)	In percentage (%)
Male	44	88%
Female	6	12%
Male: Female ratio 7.33:1		

TABLE 2: AGE DISTRIBUTION

Age Range(In years)	No: of patients(n) (Total-50)	In percentage(%)
<40	7	14%
41-60	36	72%
>60	7	14%
Mean age 51.7 years		

Out of 50 pts 44 were male and 6 were female the ratio came as 7.3:1. The mean age of presentation was 51.7 years. Smoker to non-smoker ratio is 3.54: 1.

SMOKING PATTERN

TABLE 3: SMOKER VERSUS NON SMOKER DISTRIBUTION

Smoking status	No: of patients(n) (Total-50)	In percentage(%)
Smokers	39	78%
Non smokers	11	22%
Smoker : Non smoker ratio 3.54:1		

Almost all the smokers were beedi smokers. Many were smoking both beedi and cigarette. Only very few smoked only cigarettes among smokers. More than half of non smokers were passive smokers.

COMORBID CONDITIONS

2 patients had significant past history of pulmonary tuberculosis before 10 years. 4 patients had recent history of pulmonary tuberculosis within a year and was put on antituberculosis treatment. One patient gave history of chronic obstructive pulmonary disease. Two patients gave history of other than lung cancers in their family. But none had family history of lung cancer.

SYMPTOMS AND SIGNS

TABLE 4: CLINICAL FEATURES RELATED TO PRIMARY LUNG CANCER

F/o primary lung	No: of patients(n) (Total-50)	In percentage(%)
Cough	37	74%
Dyspnoea	21	42%
Hemoptysis	14	28%
Chest pain	32	64%
E/o collapse	6	12%
E/o consolidation	4	8%

TABLE 5: CLINICAL FEATURES RELATED TO INTRATHOTACIC SPREAD

F/o intrathoracic spread	No: of patients(n) (Total-50)	In percentage(%)
Rec. laryngeal n palsy	14	28%
Pleural effusion	8	16%
SVC obstruction	5	10%
Oesophagus	6	12%

TABLE 6: CLINICAL FEATURES RELATED TO EXTRATHORACIC SPREAD

F/o extrathoracic spread	No: of patients(n) (Total-50)	In percentage(%)
Bone pain	8	16%
Brain metastasis	8	16%
Lymph node	13	26%
Cutaneous secondaries	2	4%

Among the symptoms of primary lung,the predominant symptom cough was present in 37 patients. Dyspnoea was present in 21 patients, hemoptysis in 14 patients, chest pain in 32 patients.

Among the clinical features related to intrathoracic spread, hoarseness of voice was present in 14 patients, features of pancoast tumour in 6 patients and among them horner's syndrome

in 3, then evidence of pleural effusion in 8, facial puffiness due to SVC obstruction in 5, dyspahagia due to oesophageal involvement in 6.

Among the clinical features related to extrathoracic spread, bone pain was present in 8 patients, liver secondaries in 5, evidence of CNS metastasis headache, seizures, papilloedema and focal neurological deficits were found in 8 patients, supraclavicular lymphadenopathy in 13, skin secondaries in 2.

Regarding features of paraneoplastic syndrome, 28 patients had clubbing of various grading among whom one had grade IV clubbing that was hypertrophic pulmonary osteoarthropathy.

Among the other clinical features, weight loss found in 29 patients, weakness in 16, fever in 11, other features like edema hand, chest wall mass in 11 patients.

IMAGING

TABLE 7: FINDINGS IN CT CHEST

CT chest findings	No: of patients(n) (Total-50)	In percentage(%)
Mass lesion	50	100%
Pleural involvement	20	40%
Mediastinal involvement	17	34%
Chest wall invasion	15	30%
Lung secondaries	7	14%
Phrenic nerve palsy	3	6%

TABLE 8: LOBAR DISTRIBUTION AS PER CT CHEST

Lobar distribution as per CT chest	No: of patients(n) (Total-50)	In percentage(%)
Right upper lobe	21	42%
Right middle lobe	5	10%
Right lower lobe	5	10%
Left upper lobe	17	34%
Left lower lobe	2	4%

TABLE 9: FINDINGS IN ULTRASOUND ABDOMEN

Ultrasound abdomen	No: of patients(n) (Total-50)	In percentage(%)
Normal	44	88%
Liver secondaries	5	10%
Adrenal secondaries	3	6%
Renal secondaries	1	2%
Para aortic lymph node sec	2	4%

All 50 patients had findings suggestive of malignancy in chest x-ray. The findings of computerized tomography were as follows, 1) All patients show mass lesion suggestive of malignancy and they were sorted out on the basis of site of lobe involvement 2) Pleural involvement found in 20 patients 3) Chest wall invasion found in 15 patients 4) Mediastinal involvement found in 17 patients 5) Elevated diaphragm suggestive of phrenic nerve palsy found in three patients 6) Lung secondaries from primary lung found in 7 patients. Regarding lobar involvement right upper lobe showed mass in 21 patients, right middle lobe in 5 patients, right lower lobe in 5 patients, left upper lobe in 17 patients and left lower lobe in two patients. CT brain was taken in six patients who

had clinical features of brain involvement and all showed brain metastases.

Ultrasonogram abdomen done in all patients and among them 5 showed liver secondaries, 3 showed adrenal secondaries, one showed renal secondaries and 2 showed paraaortic adenopathy.

BRONCHOSCOPY

TABLE 10: BRONCHOSCOPIC FINDINGS

Bronchoscopic findings	No: of patients(n)	In percentage (%)
Done in	30	60%
Normal	12/30	40%
Endobronchial Growth	16/30	54%
Vocal cord palsy	9/30	30%
Carina widening	4/30	13%
Tracheal invasion	1/30	3%

Fibreoptic bronchoscopy was done in 30 patients. In them 16 showed bronchial growth, 9 had vocal cord palsy, 4 had carinal widening, one had tracheal invasion and normal in 12 patients.

TYPE OF LUNG CANCER

TABLE 11: CLASSIFICATION OF LUNG CANCER

Histopathology	No: of patients(n)	In percentage(%)
Squamous cell	13	26%
Adeno carcinoma	12	24%
Large cell carcinoma	7	14%
Small cell carcinoma	4	8%
Others	16	32%

Histopathological evidence proved squamous cell carcinoma in 13 patients, adenocarcinoma in 12 patients, large cell carcinoma in seven patients, small cell carcinoma in 4 patients and carcinoma unspecified was repoted in 8 patients. And the remaining 6 had bronchoalveolar lavage examination suggestive of malignancy.

STAGING

TABLE 12: STAGING OF NON SMALL CELL LUNG CANCER

Stage of lung cancer	No: of patients(n)	In percentage (%)
I	2/46	4%
II	1/46	2%
IIIa	7/46	15%
IIIb	17/46	37%
IV	19/46	41%
Total no: of non small cell lung carcinoma patients- 46/50		

TABLE 13: DISTRIBUTION OF STAGING OF SMALL CELL LUNG CANCER

Stage of lung cancer	No: of patients(n)	In percentage (%)
Limited stage	0/4	0%
Extensive stage	4/4	100%
Total no: of small cell lung cancer patients- 4/50		

Based on the above findings staging was attempted in all patients. Among patients proved as non small cell carcinoma, carcinoma unspecified and bronchoalveolar lavage showing malignant cells two patients were in stage I, one in stage II, 7 in stage IIIa, 17 patients in stage IIIb, 19 in stage IV. Among patients proved as small cell carcinoma all 4 had extensive disease.

DISCUSSION

AGE AND SEX DISTRIBUTION

From 50 patients with newly diagnosed lung cancer, the mean age of presentation is 51.7 years compared to 54.6 years in the previous Indian study series[8] that showed that majority had clinical presentation in comparatively younger age. Young carcinoma patients (<40 yrs) constituted 14% compared to 12.5% in a madras study[11]. Sex ratio is 7.33:1 in this study compared to 5.76:1 that is an average of large series of Indian study. This implies a slightly decreased incidence in female but comparable to madras study that showed 7.9:1[11].

TABLE 14: COMPARISON OF DEMOGRAPHIC PATTERN IN LUNG CANCER

	1958-1985 Indian study series	**1986-2001 Indian study series**	**GRH study**
Male:Female	6.67:1	5.76:1	7.33: 1
Mean age (yrs)	52.16	54.6	51.7
Smoker/NonSmoker ratio	2.7:1	3-20:1	3.54:1

SMOKING PATTERN AND DISTRIBUTION

Smokers to non smokers' ratio found to be 3.54: 1 that was comparable to average of Indian study series that showed ratio ranging between 3-20:1[8].

SYMPTOMS AND SIGNS ANALYSIS

TABLE: 15- COMPARISONS OF CLINICAL FEATURES OF PRIMARY LUNG CANCER

F/o primary lung	American and European study series[15]	GRH study
Cough	8-75%	74%
Dyspnoea	3-60%	42%
Hemoptysis	6-35%	28%
Chest pain	20-49%	64%

Cough was the predominant symptom involved as reported in almost all studies. Followed by chest pain was the next predominant symptom that shows this presentation varies from western study. Probably the reason could be that Indian patients present here in advanced stage explaining the pleural and chest wall infiltration. And other symptoms hemoptysis and dyspnoea were comparable to their range.

TABLE 16: COMPARISON OF FEATURES OF INTRATHORACIC SPREAD

F/o intrathoracic spread	American and European study series[15]	GRH study
Rec. laryngeal n palsy	2-18%	28%
Pleural effusion	8-15%	16%
SVC obstruction	0-4%	10%
Oesophagus	0-2%	12%

All clinical features for intrathoracic spread at presentation here were found to be beyond the range of western study and probably the reason here again was their presentation at an advanced stage.

TABLE 17: COMPARISON OF FEATURES OF EXTRATHORACIC SPREAD

F/o extrathoracic spread	American and European study series[15]	GRH study
Bone pain	25%	16%
Brain mets	10%	16%
Lymph node	15-20%	26%
Cutaneous secondaries	1-12%	4%

Here the clinical features of brain metastasis and lymph node metastasis were higher in GRH study compared to western study. And bone pain and cutaneous were within the reference range.

COMORBID CONDITIONS

Only two patients had significant past history of tuberculois with radiological evidence. But the reports about association of tuberculosis and lung cancer were conflicting[29]. So here it is not dealt with detail. Two patients were reported with family history of other cancers and but again the reports say that the associations were also conflicting.

IMAGING

TABLE 18: COMPARISON OF DISTRIBUTION OF SITE OF LUNG CANCER

Zonal distribution	C.P.sharma et al[26]	GRH study
Upper zone	42%	62%
Middle zone	32%	20%
Lower zone	16%	14%
Entire lung	8.8%	4%

All patients had mass in chest x ray compared to Sharma et al[26] study that showed 96% mass lesion lung. Upper zone mass were found in 62% of patients compared to only 42% in his study. The predominance of upper lobe lesion is associated with smoking as the risk factor for lung cancer.

TABLE 19: COMPARISON OF SIDE OF LUNG CANCER

	N.A.Khan et al[10]	GRH study
Right lung	63%	62%
Left lung	37%	38%

The predominance of right lung involvement compared to left lung here was similar to N.A.Khan et al[10] study.

Other findings in CT such as pleural involvement was found in 28% of patients compared to 25% as showed by Behera et al[8]. Other features chest wall invasion and mediastinal involvement show the increased presentation at an advanced stage again.

All patients with clinical features of brain metastasis showed evidence of metastasis in CT brain. But as a part all patients with small cell carcinoma could not be subjected for CT brain as a routine because of poor compliance of the patient to spend due to financial constraints.

Again no patients were willing for CT abdomen as a routine procedure for staging in addition to CT chest due to the above said reason. And so they were all done ultrasonogram abdomen, looking for the evidence of liver,adrenal metastasis. Comparitively ultrasound is less sensitive to CT abdomen in detecting liver and adrenal metastasis unless otherwise the metastatic mass is large enough. So only 5 patients showed liver metastasis, 3 patients showed adrenal metastasis and one showed renal metastasis.

BRONCHOSCOPY

Out of 50 patients only 30 patients underwent bronchoscopy. Among them 53% showed bronchial growth compared to Zavala et al study that showed bronchial growth in 58%.

CLASSIFICATION OF LUNG CANCER

TABLE 20: COMPARISON OF TYPE OF LUNG CANCER BASED ON PATHOLOGY

Classification	Yang et al	Arora et al	GRH study
Squamous cell	23%	27%	26%
Adeno carcinoma	45%	21%	24%
Large cell carcinoma	35	13%	14%
Small cell carcinoma	11%	1%	8%
Carcinoma unclassified	16%	38%	32%

Non small cell carcinoma patients constituted about 64% and patients with small cell carcinoma constituted about 8%. Carcinoma unspecified constituted about 16%. Though the western studies show non small cell carcinoma accounts for 80% of lung cancer and small cell carcinoma accounts for 20% , the same did

not match with variable Indian studies including the GRH study. The reason could be multiple diagnostic techniques are needed to show a positive diagnostic yield in 70-90% of lung cancer patients. Here it was very difficult to subject the patients for multiple invasive procedures. The reason was they disagree for such procedures and it remained difficult to make them understand the pros and cons amidst of their ignorance and illiteracy. One another reason for underreporting of small cell carcinoma could be explained, was that the patients were brought with a rapid course of lung disease and they came here at an end stage. Before a complete evaluation the patients were becoming critically ill and no more investigations could be made. This sort of scenario with radiological evidence of lung cancer with rapid deterioration supports the probability of small cell lung cancer.

STAGING

TABLE 21 : COMPARISON OF STAGE OF NSCLC

Stage of lung cancer	Yang et al (Mayo clinic)	In percentage (%)
I	26%	4%
II	8%	2%
III	31%	52%
IV	35%	41%

TABLE 22 : COMPARISON OF STAGE OF SCLC

Stage of lung cancer	Yang at al (Mayo clinic)	GRH study
Limited stage	47%	0%
Extensive stage	53%	100%

72% of non small cell lung carcinoma patients were in Stage IIIb and IV. All 4 patients with small cell carcinoma were in extensive stage. This shows that most patients presented only in an advanced stage. Though surgery is the only treatment with potential for cure, there is a significant delay before patients finally present; consequently, most patients have advanced stage disease where surgery is not possible.

One of the limitations in this study was that staging procedures were done only to limited extent in all patients. In spite of it the majority showed an advanced stage.

CONCLUSION & SUMMARY

Majority of the patients here were males and majority of them were smokers, approximately 78%. There is very less awareness about hazards of smoking and this area should be highly given prior importance because in lung cancer prevention is far better than cure.

Young patients with lung cancer, age<40 years were also in higher proportion in regard to western population and all patients present at an earlier age compared to western population. These indicate the probability of air pollution contributing to lung cancer in addition to smoking. This should be taken into serious consideration and the policies regarding pollution control should be made to follow strictly.

Next to cough (72%), chest pain (64%) was the most common symptom. The general practitioners should be made aware of such symptoms and they should start evaluating the susceptible patients early for lung cancer. This would help the patient to detect lung cancer at an early stage. They should also be retrained regularly

regarding chest x ray interpretation. This could avoid possible confusion in treating the lung cancer patients as pulmonary tuberculosis based on imaging alone.

Again imaging also supported the increased smoking hazard, by showing an increased upper lobe site for lung cancer in approx. 62% of patients.

All patients were subjected only to CT thorax and ultrasonogram abdomen, and few patients did CT brain as indicated. Bronchoscopy was done in 30 out of 50 patients. The importance of complete evaluation of a lung cancer patient should be emphasized upon, including imaging apart from CT thorax, bronchoscopy and invasive procedures for a pathological proof and staging. Because this would help the patient to stage his/her disease perfect, so that he/she will be benefited by appropriate treatment.

Coming to staging majority, approx. 80% presented here in an advanced stage. This again supports the lack of awareness among public about the deadly disease and low index of suspicion

among general practitioners. As already said this should be focused upon to detect early lung cancer.

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PROFORMA

NAME AGE SEX

OCCUPATION ADDRESS

CLINICAL HISTORY:

Cough		
Weight loss		
Dyspnoea		
Chest pain		
Hemoptysis		
Bone pain		
Fever		
Weakness		
Facial puffiness		
Dysphagia		
Wheeze		
Neck swelling		
Neural pain,paresthesia		
Hoarseness of voice		
Others		

Smoking: No:of cigaratte/beedi/cherrut

No:of years

Passive smoking

Past history: PT
COPD
Diabetes mellitus
Hypertension
IHD

Family history:

GENERAL EXAMINATION	
Facial puffiness	
Clubbing	
Hypertrophic pulmonary osteoarthropathy	
Lymph nodes	
Edema	
Gynaecomastia	
Others:	

	Rt	Lt
Cervical		
Scalene		
Supraclavicular		
Others		

RESPIRATORY SYSTEM EXAMINATION

OTHER SYSTEMS

CARDIOVASCULAR SYSTEM:

ABDOMINAL EXAMN: E/o hepatic secondaries

NERVOUS SYSTEM: E/o brain secondaries
E/o spinal cord compression

OTHER SYSTEMS: E/o bone secondaries
E/o cutaneous secondaries

INVESTIGATIONS:

IMAGING	
X-ray chest PA view	
CT-thorax	
Lobes	
Nodes	
Pleura	
Mediastinum	
Diaphragm	
Pericardium & other features	
Ultrasound abdomen& pelvis : Liver Adrenals Others:	

FIBEROPTIC BRONCHOSCOPY:

Larynx
Vocal cords
Trachea
Carina
Bronchi

BIOPSY:

Cells
Nuclei

Impression

STAGING:

Tumour

Node

Metastasis

STAGE:

Note:
Recurrent laryngeal.n. palsy
Phrenic nerve palsy
Pancoast tumour
Horner syndrome
Chest wall invasion
Pleural effusion
SVC obstruction
Dysphagia
Pericardial effusion

Bone secondaries
Liver secondaries
Brain secondaries

Paraneoplastic syndrome

GLOSSARY

BAC	Bronchoalveolar carcinoma
COPD	Chronic obstructive pulmonary disease
CXR	Chest X ray
CT	Computed tomography
GRH	Government Rajaji Hospital
HPOA	Hypertrophic Pulmonary Osteoarthropathy
NSCLC	Non small cell lung carcinoma
PAA	Para aortic adenopathy
SCLC	Small cell lung carcinoma
SPN	Solitary pulmonary nodule
SIADH	Syndrome of inappropriate secretion of Antidiuretic hormone
SVCO	Superior venacaval obstruction
TTNA	Transthoracic needle aspiration
TNM	Tumour Node Metastasis

FIGURE 1. CUTANEOUS SECONDARIES IN CA LUNG

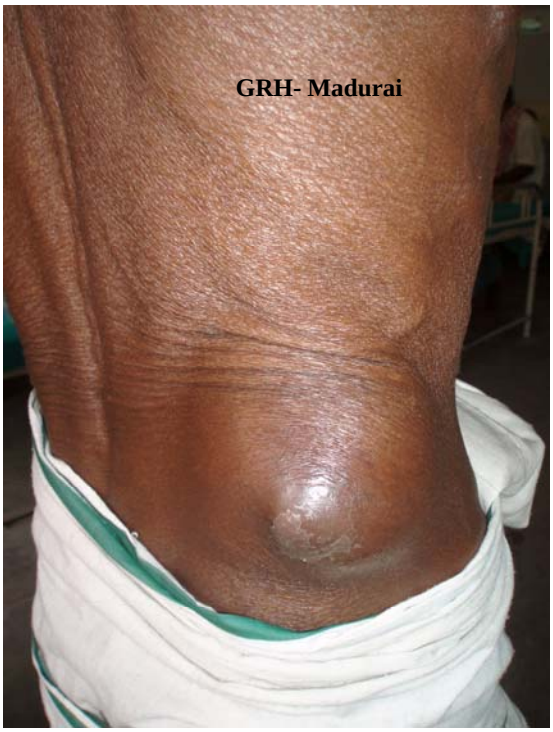


FIG.1A

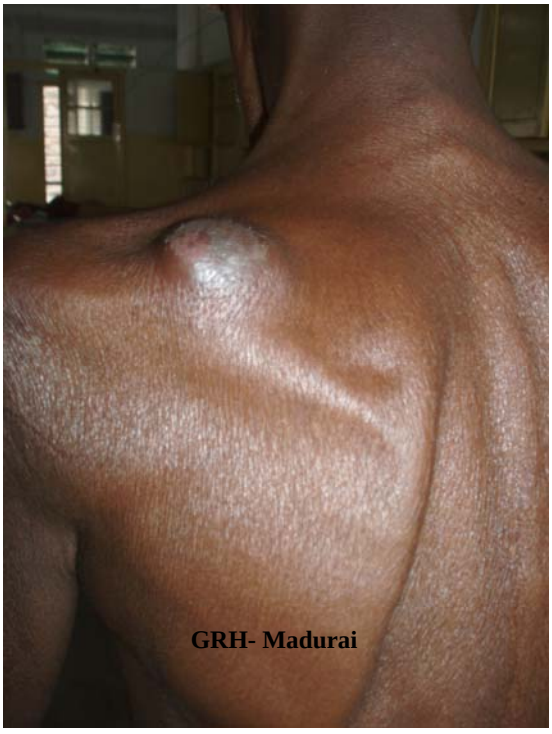


FIG.1B

FIGURE 2. GRADE IV HYPERTROPHIC PULMONARY OSTEOARTHROPATHY

FIGURE 3. BONE SECONDARIES

FIGURE 4 RIGHT UPPER LOBE LUNG CANCER

FIGURE 5. LUNG CANCER IN OLD TUBERCULOSIS

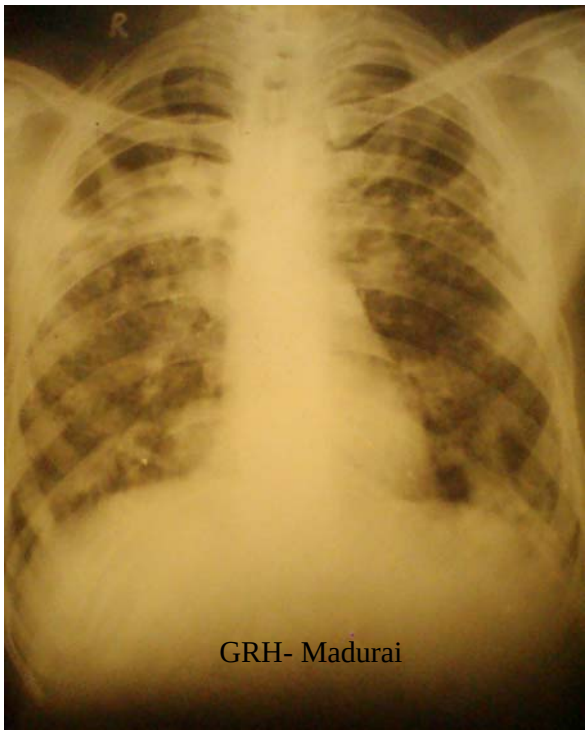


FIGURE 6. CENTRAL BRONCHOGENIC CARCINOMA WITH PLEURAL EFFUSION

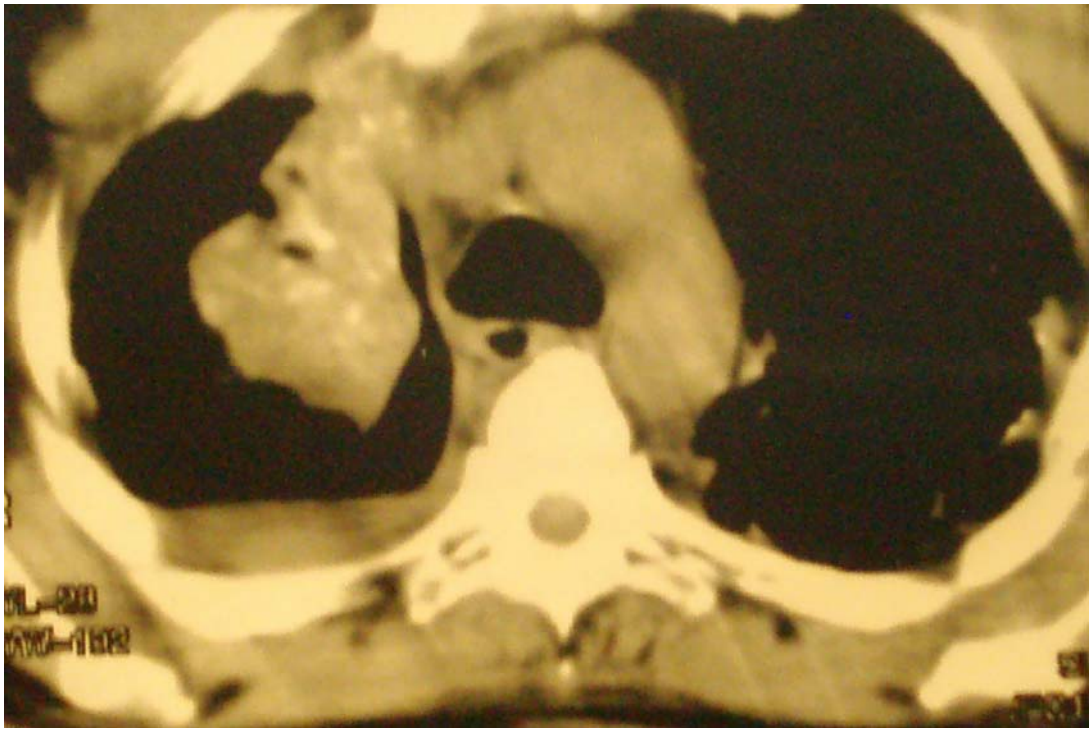
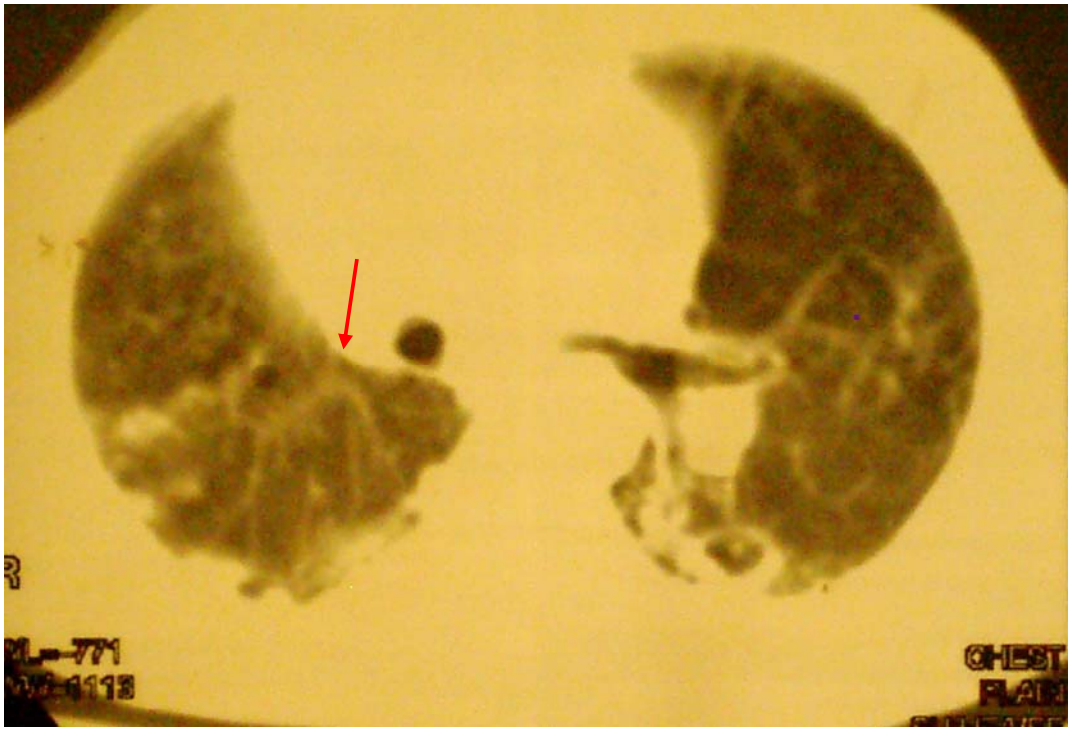


FIGURE 7. PLEURAL SECONDARY NODULES IN CA LUNG



FICURE 8. BRAIN SECONDARIES FROM LUNG CARCINOMA

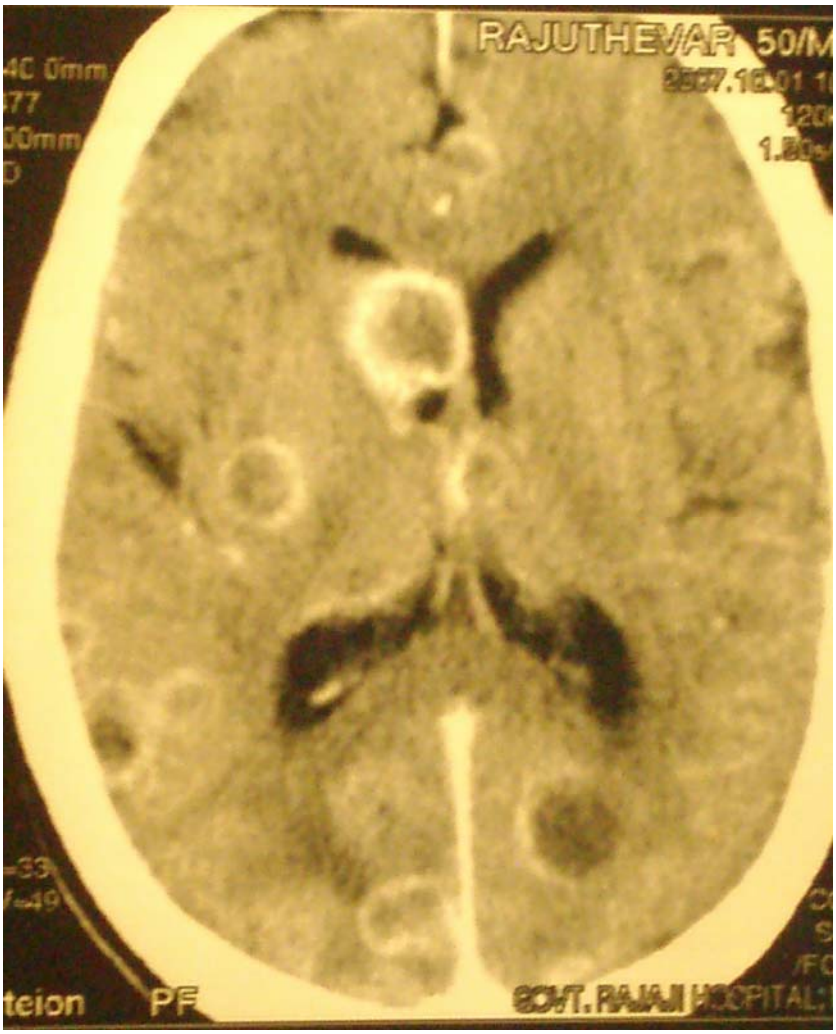


FIGURE 9. BRONCHOSCOPE

FIGURE 10. RIGHT MAIN BROCHUS SHOWING GROWTH- BRONCHOSCOPIC VIEW

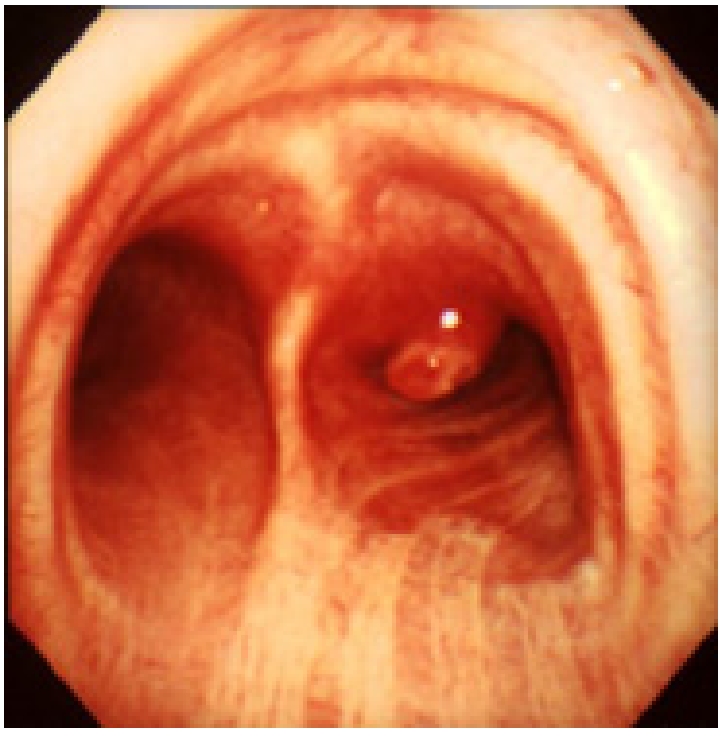


FIGURE 11

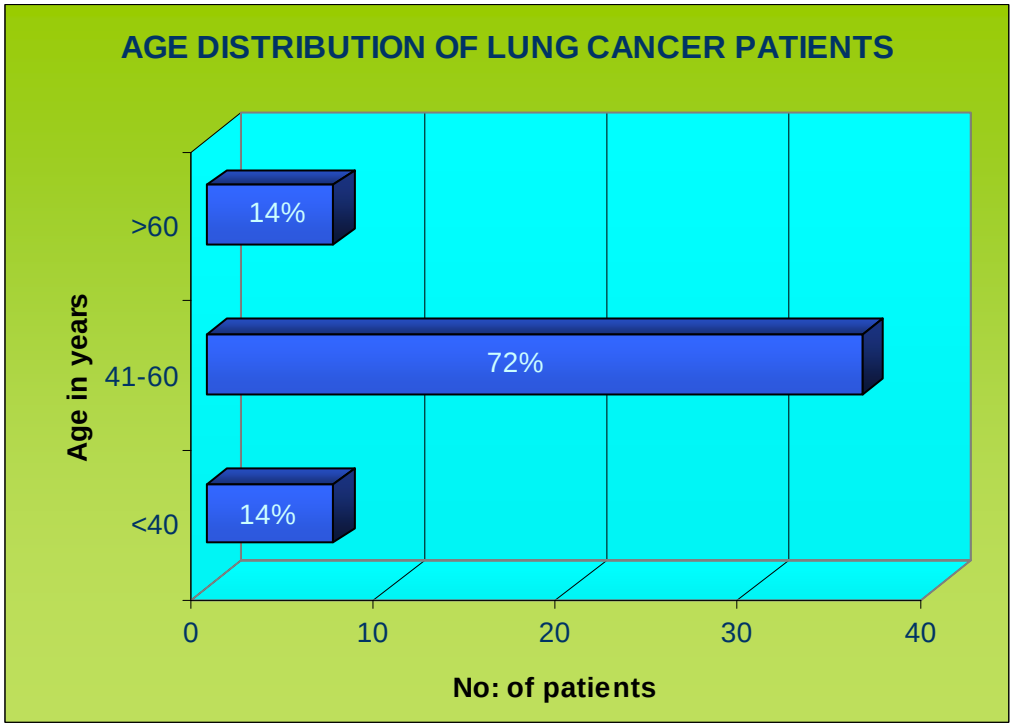


FIGURE 12

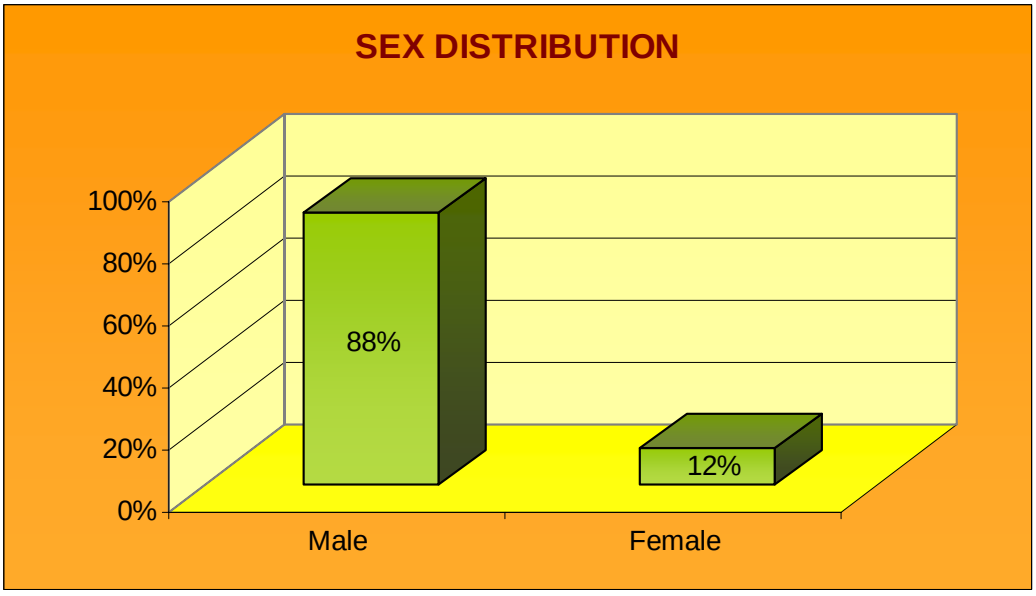


FIGURE 13

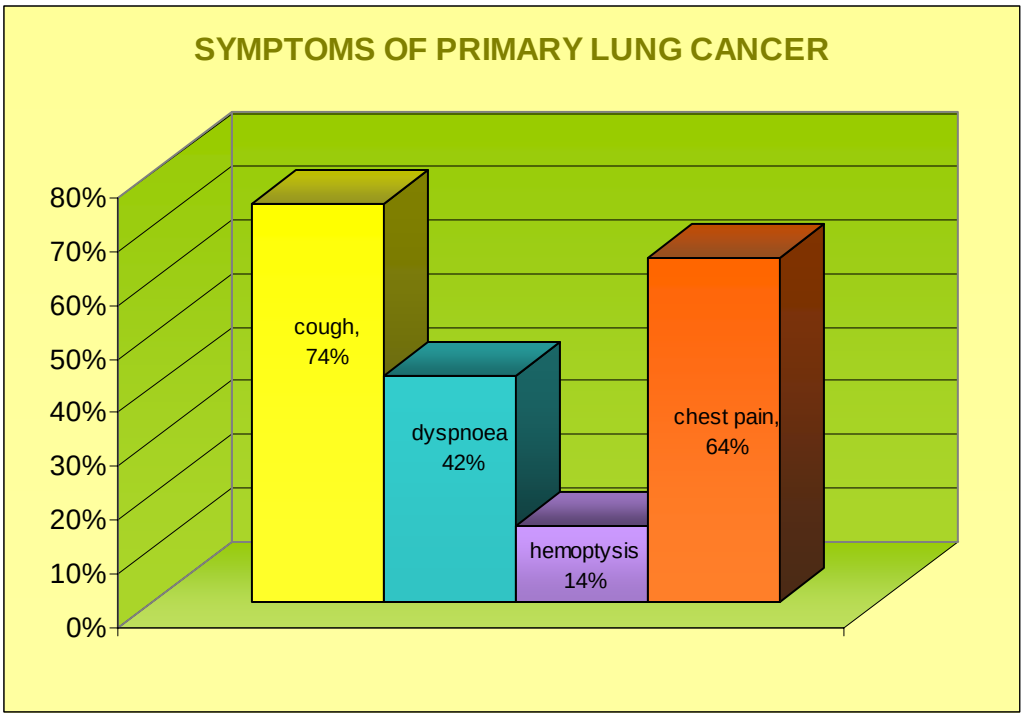


FIGURE 14

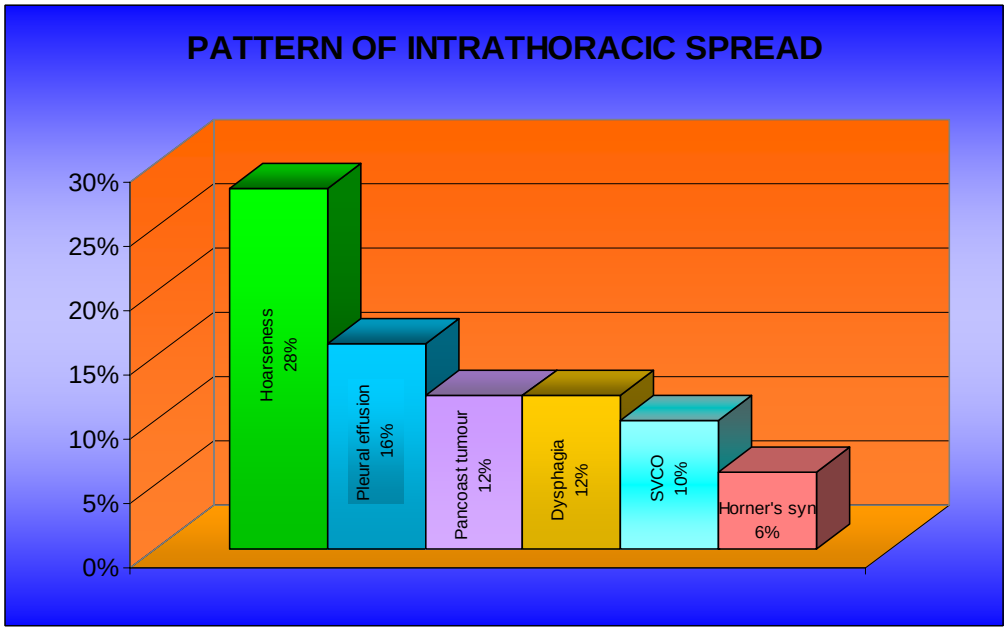


FIGURE 15

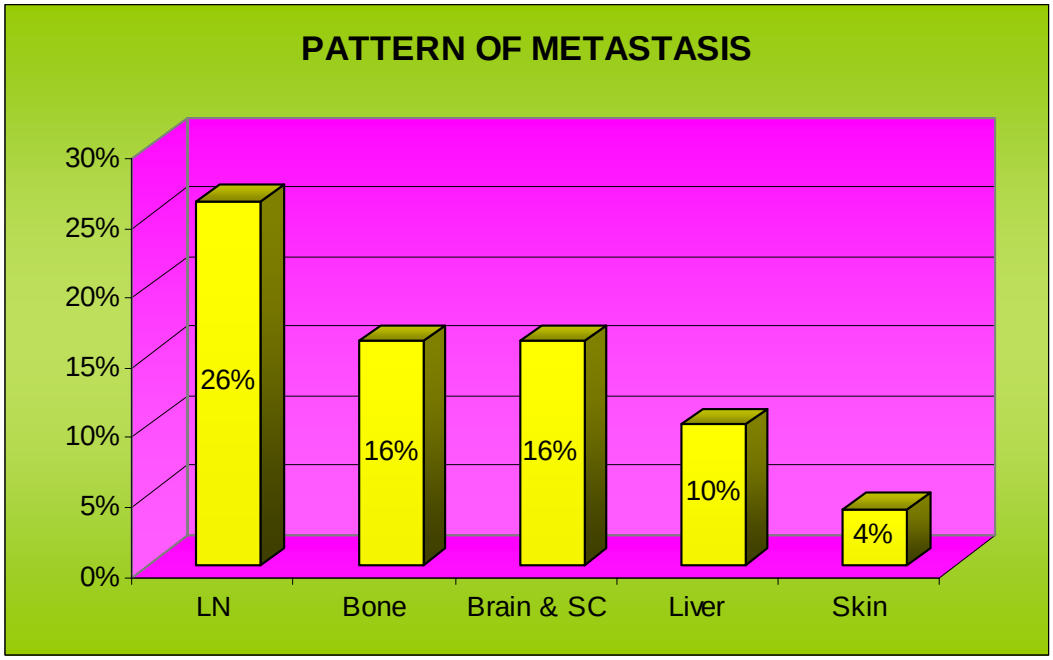


FIGURE 16

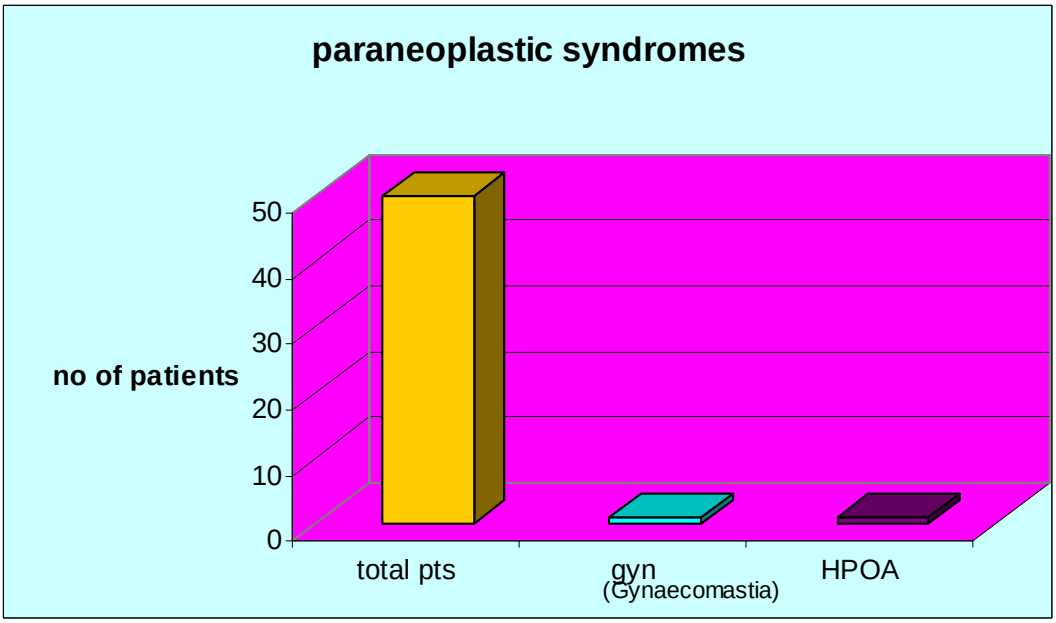


FIGURE 17

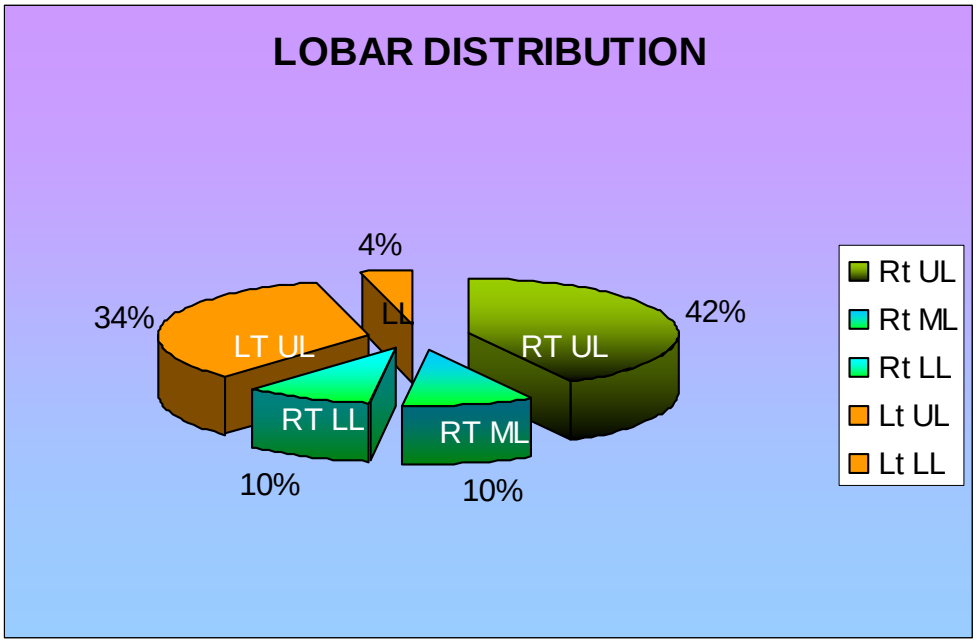


FIGURE 18

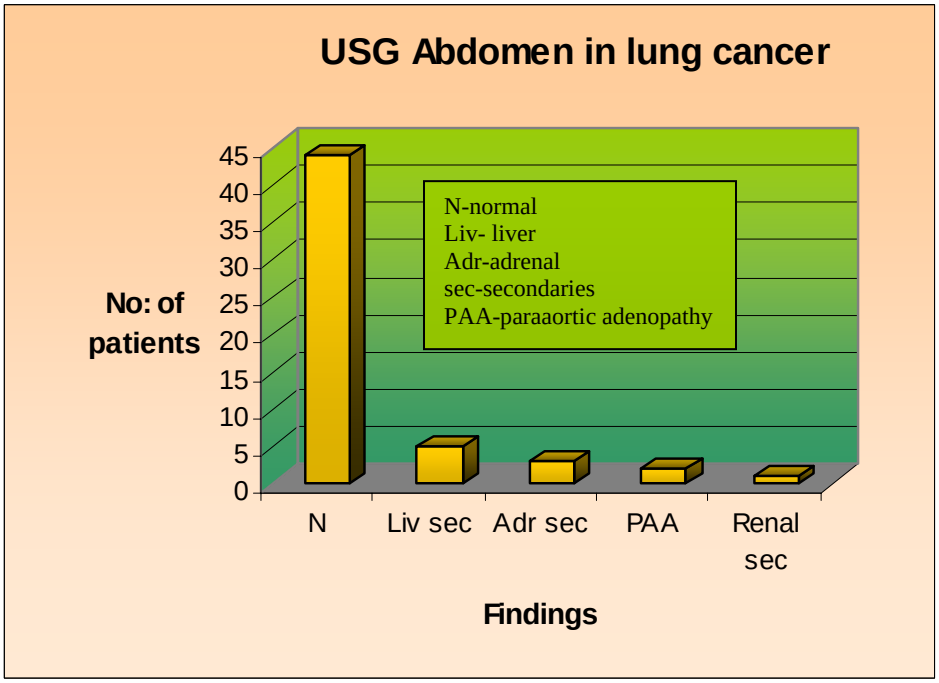


FIGURE 19

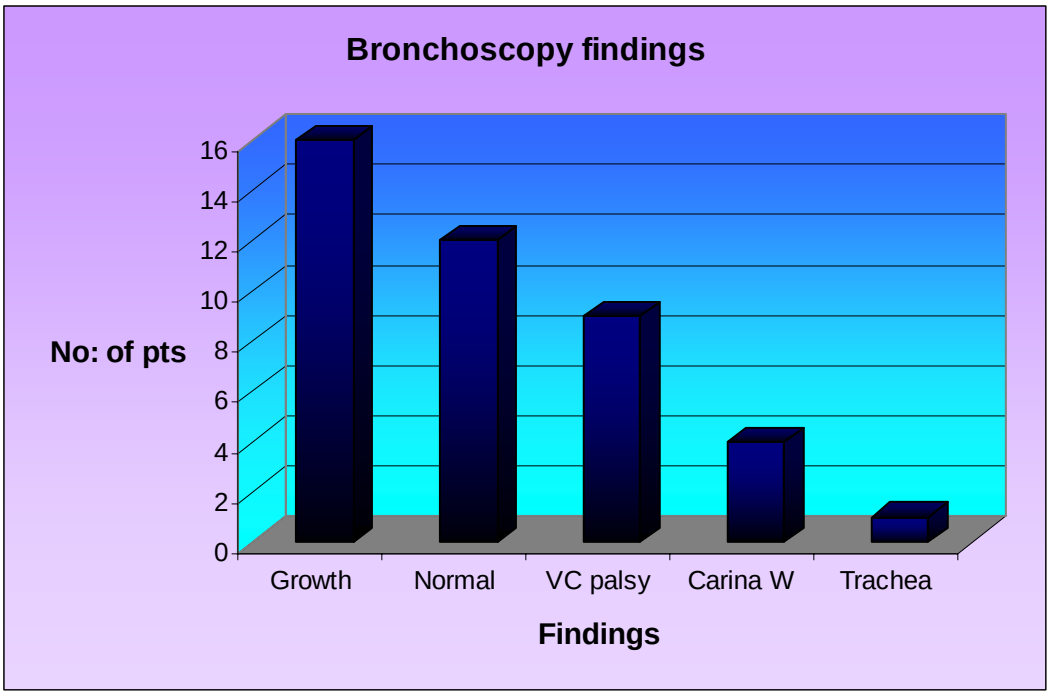


FIGURE 20

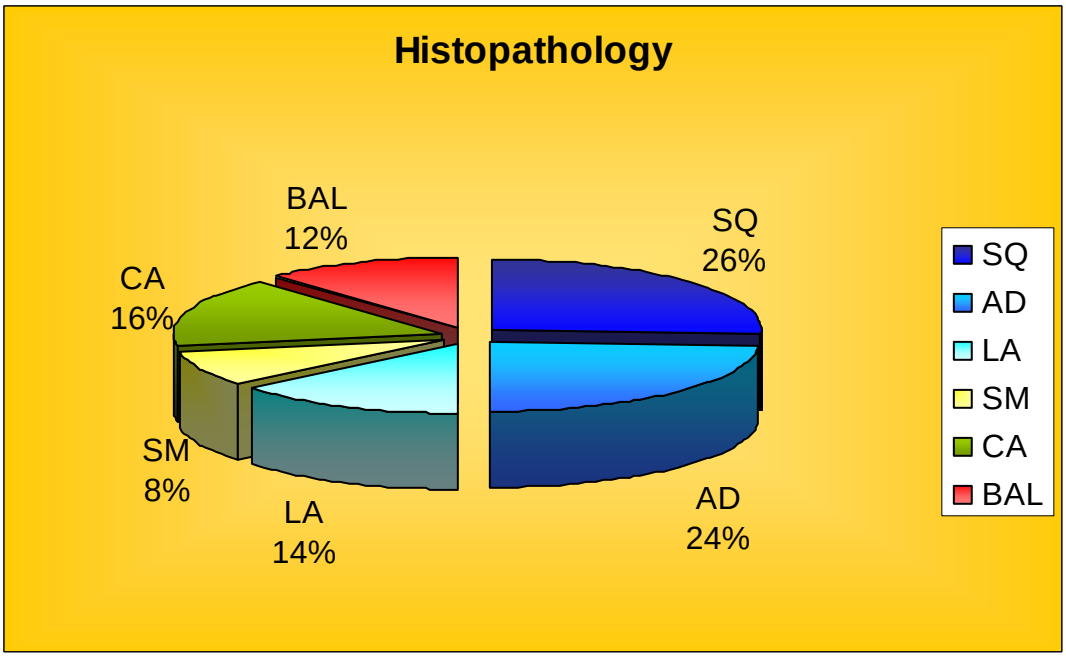


FIGURE 21

PATTERN OF DISTRIBUTION ACCORDING TO STAGE OF LUNG CANCER